Lesson learned/recommendation: In Nepal, 15% of HIV positive individuals being co-infected with Hepatitis B is a recent threat to the health authority since both the infections share a similar route of transmission which may contribute to the devastating AIDS epidemic. Hepatitis B screening should be made mandatory for all HIV sero-positive individuals.

PP-019 Intrahepatic expression of PD-1, PD-L1 and PD-L2 in Chronic Hepatitis B patients

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Background: The dysfunction of T cells may represent a mechanism of hepatitis B virus (HBV) persistence. Programmed death-1 (PD-1) and its ligands, PD-L1/PD-L2, are new members of CD28/B7 family, was reported to transfer inhibitory signal, leading to the dysfunction of T cells.
Methods and Patients: Immunohistochemical analysis of tissue samples from 56 patients with chronic hepatitis B (CHB), 12 acute hepatitis B (AHB) patients and 10 health controls was performed.
Results: PD-1 was positively expressed in inflammatory cells infiltrating the portal area dominantly. PD-L1 expression was more extensive, apart from the in portal area, also expressed on hepatocytes and sinusoidal endothelial cells. PD-L2 mostly expressed on kupffer cells (KCs) and dendritic cells (DCs) in portal area as well as interlobular. The PD-1-, PD-L1-positive cells on lymphocytes infiltrating portal area of CHB patients was $32.33\% \pm 30.56\%$ and $30.68\% \pm 27.07\%$ respectively, more than that of health controls ($2.68\% \pm 2.37\%$, $5.3\% \pm 5.62\%$ $p < 0.05$) and that of AHB patients ($12\% \pm 9.6\%$, $8.7\% \pm 7.4\%$ $p < 0.05$). The PD-L2-positive cells on KCs and DCs in CHB patients was much higher ($6.57\% \pm 7.21\%$) than that in AHB patients ($15.7\% \pm 8.2\%$) and health controls ($2.06\% \pm 2.35\%$), $p < 0.05$. According to the the severity of illness index, the PD-1, PD-L1, and PD-L2 expression rate within CHB patients was increase with the disease progression ($p < 0.05$). The expression of PD-L1 in interlobular in CHB was assayed also, in severity group the number of higher score was more than that in mild groups. And the expression of PD-1 on lymphocytes was correlated positively both with ALT ($r = 0.484$, $p < 0.05$) and AST ($r = 0.721$, $p < 0.05$), although had not apparent correlation with plasma DNA.
Conclusion: Overexpression of PD-1 and PD-L within liver might be involved in inhibiting the immune response and be a mechanism of chronicity in HBV infection.

PP-020 Hepatic steatosis is associated with host metabolic factors but not viral effect in Chinese patients with chronic hepatitis B

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Aims: To investigate the prevalence and the risk factors of hepatic steatosis in Chinese patients with chronic hepatitis B.
Methods: Patients infected with hepatitis B virus who underwent liver biopsy were included. One hundred and sixty-one patients with chronic hepatitis B were divided into two groups depending on the presence or absence of steatosis in liver biopsy specimens. The relationships of steatosis and demographic, laboratory and histological characteristics were evaluated. Liver necroinflammatory activity and fibrosis were evaluated according to the Knodell and Ishak's classification, respectively. Detection of HBsAg and HBcAg were performed in liver specimen by immunohistochemistry. Logistic regression analysis was applied to identify variables that were independently associated with the presence of steatosis.
Results: Hepatic steatosis was present in 53 patients with chronic hepatitis B (53/161, 32.9%). Steatosis was predominantly macrovesicular (49/53, 92.4%). Steatosis was associated with ages ($P < 0.001$), BMI ($P < 0.001$), fasting blood sugar ($P = 0.001$), cholesterol ($P = 0.011$) and triglyceride ($P = 0.004$). In the multivariate analysis both BMI ($P = 0.036$) and cholesterol ($P = 0.021$) were independent predictors of the presence of steatosis. No significant correlation was found with gender, ALT, AST, TBil, ALP, GGT, HBeAg, hepatitis B viral load, and histological findings between the two groups ($P > 0.05$). HBsAg and HBcAg were also detected in hepatocytes contain fat vacuoles.
Conclusion: Hepatic steatosis is present in 32.9% of patients with biopsy-proven chronic hepatitis B. Steatosis is independently associated with ages, BMI, fasting blood sugar, cholesterol and triglyceride, suggesting being as a result of metabolic factors of the host rather than the effect of viruses and it seems to have not impact the necroinflammatory activity and fibrosis.

PP-021 Effect of INF-γ treating on PD-1 expression on lymphocytes

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Background: The dysfunction of T cells may represent a mechanism of hepatitis B virus (HBV) persistence. Programmed death-1 (PD-1) and its ligands, PD-L1/PD-L2, are new members of CD28/B7 family, as co-stimulatory molecules expressing on T cells and Antigen Present Cells (APCs). Their engaging can downregulate the T cells function, including proliferation, cytokines secretion and cytotoxicity. In periphery blood, PD-1 was upregulated on virus specific-T cells, leading to the impairment of T cells. Blocking the PD-1/PD-L can improve the function of T cells.
Methods and Patients: 21 patients with chronic hepatitis B (CHB) were treated by IFN-γ (Pegintron, once a week, 0.5 or 1 mg/kg/weight). The periphery blood were taken at pretherpay, 4 weeks, 8 weeks, and 12 weeks. Periphery blood mononuclear cells were isolated from fresh heparinized blood by Ficoll-Hypaque (density:1.077 g/L) density gradient centrifugation. The PD-1 expression on lymphocytes was detected by flow cytometry (FCM).
Results: The PD-1 expression on lymphocytes at pretherapy was $14.47 \pm 5.8\%$, at 4 weeks was $9.68 \pm 3.75\%$, at 8 weeks was $6.95 \pm 2.39\%$, at 12 weeks was $6.08 \pm 1.31\%$ ($p < 0.05$).
Conclusion: Treatment of IFN-γ can downregulate the PD-1 expression on lymphocytes. Say in other words, Treatment of IFN-γ can partially restore the function of T cells.

PP-022 Clinical study of JianpiQinghua prescription combined with lamivudine to treat chronic hepatitis B

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Objective: To evaluate the antiviral efficacy of JianpiQinghua Prescription Combined with Lamivudine to treat chronic hepatitis B differentiated as syndrome type of liver depression and spleen asthenia accompanied by